Asneezia

SIR: We find Shukla's (*Journal*, May 1989, **154**, 689–690) efforts to establish asneezia as a hitherto unrecognised psychiatric symptom very difficult to accept. His earlier paper on asneezia (Shukla, 1985) had stimulated us to undertake a study at the Central Institute of Psychiatry, which has a catchment area of most of the eastern states of India. We examined 1985 consecutive new patients attending the Kraepelin Unit out-patient department. None of them spontaneously complained of asneezia. Thirteen patients reported absence of coryza or infrequent coryza. The psychiatric diagnoses of these patients were too heterogenous to be meaningful. We therefore extended our study to a GP clinic and studied 523 consecutive patients; 3.5% complained of absence of coryza and/or asneezia, either spontaneously or on specific questioning. Most of them believed that coryza or sneezing might relieve their maladies. Almost half suffered from chronic and recurrent headaches of various aetiologies; the diagnoses of the remainder varied considerably.

We do not doubt the genuineness of asneezia as a symptom, as it may occur in different population samples, but we object to a psychiatric connotation being attached to this symptom. We agree that some cultures may carry overvalued ideas regarding this symptom, but that may be true for any medical symptom.

Moreover, asneezia should be studied in a normal population first. For meaningful research to be performed, it must be operationally defined as the absence of sneezing after exposure to the most noxious inhalant allergen.

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Akathisia

SIR: The mianserin-induced restless legs in three women with major depression described by Paik *et al* (*Journal*, September 1989, **155**, 415–417) may have been due to noradrenergic-mediated inhibition of dopamine (Lipinski *et al*, 1989) lateralised to the right hemisphere (Backon & Kullock, 1989). In an analogous situation, fluoxetine-induced akathisia in four women with obsessive-compulsive disorder may have been due to serotonergic-mediated inhibition of dopamine (Lipinski *et al*, 1989). Future research needs to consider inter-relationships among diagnosis, gender, medication, and the clinical course. Metabolic rate measured by positron emission tomography may be helpful, since it is lower in the left hemisphere in major depression (Baxter *et al*, 1989), which may influence the expression of adverse effects.

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Psychosis in a transsexual

SIR: Mallet *et al* (*Journal*, August 1989, **155**, 257–259) describe a case of psychosis occurring in a male-to-female transsexual who had abruptly stopped taking oestrogens. This association is not recorded in the literature. A patient of mine, however, had a similar history.

Case report. The patient, now aged 31 years, is an articulate male-to-female transsexual. He has been cross-dressing from the age of 15, and has lived as a woman for the past 3–4 years. He had had oestrogens intermittently for some 10 years, and then continuously for 3 years until he reduced them substantially in January 1987.

He came to our notice for having, in May of that year, set fire to a house believing it to be full of evil spirits. A history was obtained of his developing a paranoid psychosis dating from that January, characterised by delusions of persecution, and a belief that the TV was talking to him and that his thoughts were being interfered with and controlled. He felt that he was involved in a spiritual struggle, and that his friend Roger was actually Roger's *doppelgänger*.

He was remanded in custody and, in the prison hospital, accepted antipsychotic medication, with a rapid improvement in his condition and loss of all psychotic symptoms.

His grandmother had died in early January, and in March his brother had been injured in the Zeebrugge ferry disaster. He was also under stress having been estranged from his family. However, the patient was convinced at the time that the reduction in oestrogens was the principal factor. He had come to this conclusion because at the age of 26

he had reduced his oestrogens and had experienced similar, although less severe, psychotic symptoms which eventually remitted at the same time as he increased the oestrogens.

Clearly a case history like this proves little, but the similarity to the case described by Dr Mallet *et al* is interesting.

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Fluvoxamine and lithium: an unusual interaction

SIR: The Committee on Safety in Medicines (1989) have reported the increased incidence of 5HT-related side-effects, from hyperarousal and nausea to tremor and convulsions, occurring with the combination of fluvoxamine and lithium.

We report a case of irresistible somnolence, the first time such a side-effect has been reported.

Case report: A 39-year-old married woman with a history of previous bipolar affective swings was admitted with depressed mood, biological features of depression, and second person, mood congruent, auditory hallucinations. She had been off all psychotropic medication for 10 months prior to the current episode. She was started on fluvoxamine, with good effect.

Because of the disruptive effect of the mood swings on her family life, it was decided to start treatment with lithium. Lithium carbonate (slow-release, 400 mg nocte) was added, and the patient went on weekend leave the following day.

On her return to the ward she was in a somnolent state, rouseable with some difficulty and falling asleep again almost immediately. Her husband reported that this condition had been continuous after the first night of her leave.

Neurological examination was normal apart from the level of consciousness. Lithium level (20 hours after the last dose) was 0.2 mmol/l; full blood count, urea and electrolytes, and liver function tests were all normal.

All medication was stopped. The following day she was fully conscious and becoming mildly elated. Lithium (800 mg nocte) was restarted 10 days later, and a satisfactory serum level achieved. She remains well on this medication.

After recovery she described the sleep as peaceful, refreshing, and untroubled by dreams. There was no sleep paralysis or cataplexy. An EEG was not performed, as she recovered on withdrawal of medication.

Fluvoxamine causes increased synaptic levels of 5HT. Lithium potentiates this effect by increasing 5HT synthesis (Gillies *et al*, 1986). In combination, therefore, one would expect these drugs to produce increased incidence and severity of 5HT-related side-effects.

Various studies have given equivocal or contradictory results as to whether somnolence can be directly caused by increased 5HT concentrations in the brain (Parkes, 1985).

Our patient had been treated with fluvoxamine alone for a previous depressive episode, and lithium alone on recovery from this episode, with no reported side-effects. We suggest that the combination of these two drugs caused the reported somnolence, possibly by increasing the 5HT levels in the brain to toxic levels, or by an idiosyncratic effect in this particular patient.

Combined therapy of lithium with an antidepressant is a recognised combination. Further case reports will elucidate whether this was an idiosyncratic reaction or a direct interaction of these two drugs.

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Fluvoxamine and liver enzymes

SIR: *Case report:* A 74-year-old woman with depressive psychosis was transferred to a psychiatric ward from a medical ward where she had been admitted for acute constipation. Systemic investigation at the time revealed a marked kyphosis, hypertension, a nodular swelling of the thyroid gland, and severe constipation. Routine blood tests, thyroid and liver function tests, chest X-ray, ECG, and computerised tomography scan were all normal. Family history was unremarkable; past medical illnesses included hysterectomy at the age of 25, hypertension, and left cataract operations.

Amitriptyline was prescribed, but stopped after she developed acute congestive cardiac failure, which responded to frusemide, amiloride, and nifedipine. On recovery, she was treated with fluvoxamine (50 mg nocte, increased to 75 mg after a week, and 100 mg after a fortnight). Prior to starting treatment, a series of four blood tests showed normal gamma-glutamyltransferase (γ-GT) and alkaline phosphatase (ALP), and marginally raised bilirubin on one occasion. Four days after initiation of fluvoxamine, ALP increased to 423 IU/l (normal <330 IU/l) and bilirubin increased to 31 IU/l (normal <22 IU/l). Weekly blood tests were undertaken, but fluvoxamine stopped after 3 weeks due to persistently raised ALP and bilirubin. ALP levels were highly significantly increased during treatment